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Pneumococcal Endocarditis

Pneumococcal endocarditis is a septic complication of acute pneumococcal infections—most commonly pneumococcal pneumonia. The introduction of antimicrobial agents of both synthetic and natural origin during the past 20 years has been responsible for a dramatic reduction in mortality from the pneumonococcal pneumonias; also, these agents have reduced the incidence of purulent complications of these pneumonias and, in many instances, have been responsible for the cure of complications which at one time were considered almost universally fatal. In view of the fact that agents are available for the treatment of such complications, a review of the literature on pneumococcal endocarditis was made in the hope that a better appreciation of the clinical features of the disease entity might be made available, thereby permitting therapy to be instituted earlier.

Pneumococcal endocarditis has been reported among all age groups. While there have been several case reports of patients in their first as well as ninth decades, the median age incidence lies in the fifth decade. Neither sex nor race seems to have a predisposing influence on the incidence of pneumococcal endocarditis. The complication was noted much oftener in males than in females, but likewise the primary disease, pneumococcal pneumonia, is seen more frequently in males. Similarly, pneumonia is seen in large hospitals of the north temperate zone more often in Negroes, although the incidence of endocarditis is no greater among Negroes than among white pneumonia patients.

While pneumococcal endocarditis may follow any primary pneumococcal infection, the overwhelming majority of reported cases followed pneumococcal pneumonia, usually of the classical lobar type. Less frequently, the pneumonia was of the lobular type or may have been diagnosed only at necropsy. In the rare instance, the portal of entry was difficult to ascertain; endocarditis due to a pneumococcus has been reported to follow meningitis, mastoiditis, tooth extraction, septic abortion, urethral dilatation, and surgical intervention for various conditions.

That antecedent disease of the valvular endocardium predisposes to attack by the pneumococcus is doubtful. In the present series of cases collected from the literature, a history of rheumatic heart disease or physical signs of valvular damage was noted in less than 10% of cases. On the other hand, many more cases were shown at necropsy to have histological stigmata of rheumatic fever. It may be safely concluded that there does not appear to be nearly so great a predisposition of antecedent valvular damage to attack by the pneumococcus as has been shown to be true in the case of Streptococcus viridans and related groups of streptococci. While an occasional case of pneumococcal endocarditis has been reported as being engrafted on a valve damaged by syphilis, this complication happens rarely.

In view of the fact that more than 90% of the cases of pneumococcal endocarditis represent complications of pneumococcal pneumonia, the clinical

picture of the complication may be completely masked by the primary disease. While classical pneumococcal pneumonia may affect people of all ages, endocarditis is primarily a complication affecting patients of middle age and older. The complication is frequently ushered in by a sudden chill early in convalescence at which time relapse, recrudescence, or inadequate therapy may offer a plausible explanation to the physician for the chill and subsequent fever. At other times, the complication apparently has its onset in the febrile course of the patient and may thereby escape detection. In another group of patients, the onset of the endocarditis came late in the course of the disease or even after recovery from a presumably trivial respiratory infection; during the era of serum therapy, the exacerbation of fever frequently required differentiation from serum sickness as an explanation therefor.

Although the patient may satisfactorily tolerate the chill and subsequent fever, there is usually an increase in the anxious appearance of the patient with an endocardial complication of pneumonia. Frequently, the evidence of increased toxemia is the principal sign to alert the physician to the diagnosis when no localizing signs are present.

The course of patients with pneumococcal endocarditis is usually one leading to a rapidly fatal outcome. These patients usually become increasingly toxic and succumb either to toxemia or meningitis. Some expire because of cardiac failure secondary to deformity or rupture of a valve cusp. The duration of the disease varies considerably even in untreated patients. It is difficult to ascertain the precise onset of the endocarditis, especially in patients whose endocardial lesion merges with the primary pulmonary disease. As a rule, these patients do not survive longer than 6 weeks; notable exceptions to this survival period have been reported.

Therefore, it may be said that the diagnosis of pneumococcal endocarditis must be based primarily on a high degree of suspicion. The persistence of pneumococcemia or the recurrence of a positive blood culture of the pneumococcus in a patient receiving antimicrobial therapy directed at this organism should alert the physician to the possibility that endocarditis may be present. If laboratory facilities are available which detect not only the pneumococcus but also simultaneous circulating type-specific antibodies, the suspicion should certainly be increased; this is especially true when no other pyemic focus can be ascertained. Moreover, if embolic phenomena occur in the course of the patient's illness, the likelihood of endocarditis is increased. When heart murmurs develop or change in intensity—especially those heard in diastole—they can usually be ascribed to endocarditis in the face of a persisting pneumococcemia.

Pneumococcal endocarditis remains a complication of serious prognostic import. Inasmuch as it may occur in patients with previously undamaged valves, every pneumococcal infection should be treated vigorously and early with the aim of preventing endocardial complications. (Ruegsegger, J. M., Pneumococcal Endocarditis: Am. Heart J., 56: 867-875, December 1958)

Bronchogenic Carcinoma

Although many studies have been made and figures compiled of bronchogenic carcinoma, relatively few concerning the individual cell type have been reported. In the treatment of most tumors, one of the factors concerning the ultimate prognosis of the disease is its cell type. With bronchogenic carcinoma, however, the studies concerning individual cell types—although relatively few—have been largely controversial.

Many authors hold the view that the cell type is a highly valuable prognostic element. Others have reported that the individual cell type has no prognostic significance. Although at present there appears to be more evidence favoring a varied clinical course dependent on the different cell types, it is readily apparent that there is no complete agreement on this point. This study is concerned with this problem of correlating the clinicopathologic features of the various cell types.

The clinical material was composed of all histologically proved cases of bronchogenic carcinoma treated at the Johns Hopkins Hospital, 1942 through 1951. No patient after 1951 was included in order to insure that all patients would have been followed at least 5 years. The histologic material from these patients was from either surgical or autopsy specimens. The clinical records were studied and the data recorded. Separately, and without knowledge of the clinical history, all histologic specimens were examined and the cell type was determined. As far as could be ascertained, all cases in which there was another primary cancer were eliminated in order to prevent including metastatic tumors of the lung.

During this 10-year period, there were 351 histologically proved cases of bronchogenic carcinoma. Histologic diagnosis was made from tissue taken at thoracotomy alone in 126 cases. In 64 cases the diagnosis was made histologically at autopsy, and in 56 cases the diagnosis was made from material obtained at bronchoscopy. There were 43 cases diagnosed from lymph node biopsy and 5 from cell block study only. The remaining 57 cases were diagnosed by combinations of the above methods. Histologic material in some cases was limited to cytologic studies and bronchoscopic biopsies both of which occasionally were inadequate to make a cell-type diagnosis, and no diagnosis other than bronchogenic carcinoma could be made. In other instances—even in resection cases—no diagnosis of cell type could be made with any degree of accuracy because of lack of a definite cellular pattern. These cases made up a group of 18 (5%). The remaining 333 cases (95%) were diagnosed as to distinct cell type.

Admittedly, it is difficult to classify various histologic patterns in any tumor, and indeed, in many instances it is entirely unwarranted. One definitely should not try to force any tumor into a certain classification for the sole purpose of classification. There are many tumors which cannot be classified as to specific cell type with any degree of reliability; continual subgrouping or subclassification adds nothing of value, serving only to make

the issue more confusing and less accurate. However, most tumors in this study (95%) were of a predominant cell type. These cellular patterns are squamous-cell carcinoma, adenocarcinoma, and small-cell and large-undifferentiated-cell types.

Of the 333 histologically typed cases out of the 351 in the present study, 127 patients received no surgical therapy. Most of the patients had evidence of metastases and their tumors were clearly inoperable; a few had other unrelated conditions of such a severe nature that operative therapy was thought to be unwise; a few patients refused surgical treatment. There were 206 patients who underwent thoracotomy, but only 107 had some type of resection. Therefore, although the operability rate was 62%, the resectability rate dropped to 32%.

Striking differences existed in the ability to treat patients having the various cell types. There were 111 patients with squamous-cell carcinoma who underwent thoracotomy; of these, 71 had resectable tumor. This was an operability rate of 74% and a resectability rate of 47%. There were 11 palliative resections, and 60 resections were done with the idea of curing the patient. Of the 40 patients with adenocarcinoma, 16 had inoperable lesions. In the remaining 24, the tumors were operable but only 11 were resectable. Of these resections, 1 was palliative and the other 10 were done as a curative measure. This gives an operability rate of 60% and a resectability rate of 28%. Of the 95 patients with large-cell carcinoma, 44 had inoperable tumors. The remaining 51 tumors were operable, 23 being resectable. This was an operability rate of 54% and a resectability rate of 24%. Less than one-half of the patients with the small-cell type had operable carcinoma, only 20 out of 48 having had thoracotomy. Of these, only 2 had resections. This was an operability rate of 42%, but a resectability rate of only 4%. Thus, at one extreme, the squamous-cell type was resectable in 47%, whereas, at the other extreme, the small-cell type was resectable in only 4%. Of the patients with resections, the hilar lymph nodes contained tumor in these percentages: squamous-cell carcinoma, 27; small-cell carcinoma, 50; adenocarcinoma, 55; and large-cell carcinoma, 56.

Follow-up data on all patients in this study were complete in 95%; there was a 99% follow-up of the patients who had had resection. Nineteen patients survived 5 years or longer after resection. This is an over all survival rate of 6% of all patients entering the hospital and a survival rate of 18% of all patients undergoing resection.

It is obvious that information compiled from a group of patients having a tumor of a specific cell type cannot be directly applied to any one given patient with a tumor of the same cell type. The fact remains that in any given patient the factors of importance are the exact location of the tumor and to what extent and by what route spread has occurred. Therefore, to conclude that, because the patient has a particular type of tumor, his prognosis will be the same as that for the group is not sound. However, it can be stated that the course of the various cell types tends to fall into a more or less definite

pattern which gives valuable information. Thus, in dealing with a patient having a small-round-cell carcinoma there is very little chance of effecting a permanent cure. In this group, not a single patient was cured. On the other hand, in dealing with a patient having squamous-cell carcinoma, the chances are much more favorable—considering that 10% of all patients who entered the hospital with this type of cancer and 27% of those who left the hospital after resection survived 5 years or longer. Between these two extremes in prognosis are patients with adenocarcinoma and the large-cell type.

This fact does not mean that the patient with the small-cell type should not be operated upon if there are no surgical indications of inoperability; however, because of its location and rapid growth and spread, very few of such tumors will be resectable at the time of operation. In this study, only 4% of all tumors of this type were resectable. These statistics do not indicate that the small-cell type of carcinoma having a favorable location should not be resected, but rather point out that the chance of finding a favorable lesion in these cases is extremely small and that any heroic measure to attempt a cure in patients with this type of tumor is probably useless. On the other hand, not all patients with squamous-cell carcinoma will have a resectable tumor, but the chance of finding a favorable lesion is relatively good because almost one-half of the patients entering the hospital with this cell type of tumor were able to have resectional therapy. More of the patients with adenocarcinoma and the large-cell types of tumor will be cured than those with the small-cell type. However, even in these groups there is a considerably lower percentage surviving than in the group of patients with squamous-cell carcinoma. It would, therefore, appear that prognosis cannot be founded on the cell type alone, but that the cell type is one important factor in the prognosis because it determines to a large extent the location, rate of growth and spread and, therefore, resectability and cure. (Collins, N. P., Bronchogenic Carcinoma - Importance of the Cell Type: Arch Surg., 77: 925-932, December 1958)

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Pulmonary Infiltration

The significance of roentgenographically unchanging pulmonary lesions, in the absence of bacteriologic or pathologic proof of etiology, is difficult to determine. It has been shown by Britten that many such cases subsequently develop tuberculosis. The problem of the diagnosis of these cases is of special importance to the Armed Forces where intimate environmental situations exist. Therefore, it became necessary to assess accurately the risk of such persons developing active pulmonary tuberculosis and thereby becoming sources of infection.

The purpose of this study was to measure the risk among military personnel retained on active duty after clinical study had rendered diagnoses of "infiltration, pulmonary, cause undetermined" or "fibrosis, pulmonary, cause undetermined." These two diagnoses were established in 1948 by a Joint Armed Forces decision in order to afford an orderly classification of unchanging, predominantly non-calcified pulmonary lesions of unknown etiology.

A study group consisting of personnel with the above diagnoses and a control group consisting of personnel diagnosed as having "hemorrhoids" were utilized to determine the risk of tuberculous infection in the two groups, i.e., the incidence rates of tuberculosis.

Both the study and control groups consisted of active duty Naval and Marine Corps personnel who had been admitted to naval medical facilities, clinically studied, and returned to duty during 1951. In order to make the results of the study meaningful, all cases who had had a diagnosis of active tuberculosis during or prior to 1951 were excluded. Also, to determine how many of the remaining personnel developed pulmonary tuberculosis between 1952 and the end of 1954, all cases were excluded who had left the service during 1951 for any reason, e.g., release from active duty, discharge for medical reasons, or death.

There were 268 cases with the diagnosis of "infiltration, pulmonary, cause undetermined" or "fibrosis, pulmonary, cause undetermined" who met the above criteria and were, therefore, included in the study group. The control group consisted of 493 Naval and Marine Corps personnel, also meeting the above criteria who were admitted and returned to duty in 1951 with the diagnosis of "hemorrhoids." Members of the control group were selected by means of systematic sampling of the 3436 such cases occurring in 1951.

Cases with the diagnosis of "hemorrhoids" were chosen as the control group because there is no known relationship between hemorrhoids and tuberculosis and because the over all age distribution of these cases was similar to that of the study group.

The percentage of non-Caucasians was slightly higher in the study than in the control group (7.5% as compared to 4.3%). This difference, however, was found to be not statistically significant. The 493 persons in the control group contributed 16,073 man-months of exposure while in the study. During

that time, no members of the control group developed active pulmonary tuberculosis.

The 268 persons in the study group contributed 8842 man-months of exposure. Sixteen members of this group developed active pulmonary tuberculosis during the period of the study, yielding a rate of 1.8 cases per 1000 man-months of observation. Of these 16 cases, 13 were Caucasian and 3 were non-Caucasian. With respect to age, 10 were 30 years of age or older in 1951.

A review of the x-rays of personnel included in the study group revealed pulmonary lesions which were localized and contained little or no calcification. In general, no radiologic difference could be seen between lesions diagnosed as "fibrosis, pulmonary, cause undetermined" and those diagnosed as "infiltration, pulmonary, cause undetermined."

As shown, the members of the study and control groups consisted of active duty Naval and Marine Corps personnel with approximately the same age and race distribution, who were admitted to naval medical facilities and returned to duty in 1951. None had a prior history of active tuberculosis. The only gross variable in the two groups, therefore, was the diagnosis with which they were returned to duty. All members of the study group had been discharged from the hospital with a diagnosis representing an "inactive" predominantly noncalcified pulmonary lesion of unknown etiology, while all in the control group had been returned to duty with the diagnosis of "hemorrhoids."

If the presence of the pulmonary lesions of the study group bore no relationship to pulmonary tuberculosis, one would expect that the incidence of tuberculosis in the study group would be very nearly the same as in the control group. When, however, the actual tuberculosis incidence of 1.8 cases per 1000 man-months in the study group is applied to the 16,073 man-months of observation contributed by the control group during the years of study, the expected incidence of cases of active tuberculosis in the control group is 29.1, whereas the actual observed incidence was zero.

Examination of Navy Statistics for the years 1952, 1953, and 1954 revealed 1206 new cases of active pulmonary tuberculosis developing in a population experiencing 36,800,000 man-months of exposure. Applying the study group attack rate of 1.8 cases per 1000 man-months contributed by the U.S. Navy during the study, there is an expected incidence of 66,590 cases. The actual incidence of 1206 new cases, therefore, represented only 1.8% of the expected cases.

In a recent study by Wier and Tempel, relapses occurred in 4.1% of a selected group of military personnel with active pulmonary tuberculosis who had been treated to the arrested stage and returned to duty by the U.S. Army. Analysis of this group was made for those who had been returned to duty between 1952 and 1955. Tuberculosis occurred in 5.9% of the pulmonary infiltration and fibrosis cases in the present study group. While the

Army group is not completely comparable to the present study group for the purpose of statistical analysis, it is interesting to note that the risk of developing active pulmonary tuberculosis in this group may have been as great or greater than the risk of relapse in the selected Army personnel who had been returned to duty after their tuberculosis had been treated to the arrested stage.

Britten determined that, of 79 Naval and Marine Corps personnel returned to duty after clinical study in 1949 with diagnoses of pulmonary infiltration or fibrosis, 9 developed pulmonary tuberculosis during the next 3 years. This present study supports these findings which are suggestive of a very high incidence of active pulmonary tuberculosis among such cases.

In view of the demonstrated frequent development of active pulmonary tuberculosis in persons "proven" by clinical study to have predominantly noncalcified "inactive" pulmonary lesions of unknown etiology, the often held concept of the lack of clinical significance of such lesions becomes questionable. In all cases in this study developing active pulmonary tuberculosis, the disease was seen to exist in the area of the lung previously evaluated. In several cases, the diagnosis of active tuberculosis was made within 8 months after the 1951 hospitalization. These facts make it reasonable to assume that many of these cases were actually "missed tuberculosis" in which the diagnosis could not have been definitely made by means of present clinical and laboratory methods.

Examination of the clinical records revealed that in every case there had been a thorough evaluation made during the 1951 hospital admission. Incomplete clinical evaluation did not seem, therefore, to account for the abnormally high rate of development of tuberculosis. That it developed at such a high rate, indeed, points up the inadequacy of present diagnostic armamentarium and, until it is improved, the only means of early detection of tuberculous activity in such cases will continue to be frequent follow-up examinations over a prolonged period of time. To insure adequate follow-up, the establishment of a standardized procedure of examination and a central registry of case reports appears to be desirable.

Because pulmonary tuberculosis is difficult to diagnose in the absence of symptoms and bacteriologic confirmation, there is reluctance on the part of many physicians to make the clinical diagnosis under these conditions. This is understandable, but this study shows that if tuberculous activity is to be detected early and its dissemination kept to a minimum, the physician's index of suspicion must be high in the case of the "stable" predominantly noncalcified pulmonary lesion.

It is concluded that persons in the U. S. Navy, determined by means of present diagnostic methods to have either pulmonary infiltration or fibrosis of unknown etiology, are more likely to have or to develop active pulmonary tuberculosis than is now generally appreciated. (Captain J. F. Chace MC USN, Rockoff, S. D., M. D., Hellman, L. P., B. S., M. A., Pulmonary

Infiltration and Fibrosis of Unknown Etiology - The Risk of Developing Active Pulmonary Tuberculosis: Arch. Int. Med., 102: 367-374, September 1958)

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Cardiac Asthma

Cardiac asthma is thought to occur in about 8% of cases of organic heart disease. The present report develops the thesis that the management of cardiac asthma requires in most instances the disciplines of the cardiologist and the allergist. To do this, current concepts of cardiac asthma are presented from the points of view of the cardiologist and the allergist, and the problems of diagnosis and management are outlined.

McGinn and White provided the following vivid description: "Cardiac asthma is a name applied to a kind of dyspnea peculiar to organic cardiac disease. For this particular condition, it is distinctive and preferable to such other terms as pulmonary edema or paroxysmal dyspnea because it is truly asthmatic in nature and is fundamentally of cardiac origin. Cardiac asthma is paroxysmal, coming on usually in sleep, but at times following exertion. An attack quickly rises to a peak, is accompanied by both inspiratory and expiratory difficulty and frequently by a terrifying sense of suffocation which causes the patient to sit up or to stand erect and even to go to the window for air. The attacks last from a few minutes to a few hours averaging about an hour and leave the patient in an exhausted condition for hours or days."

Cardiac asthma is precipitated by acute failure of the left ventricle. It is a common feature of syphilitic and hypertensive cardiovascular disease, aortic valvular disease, coronary insufficiency, and myocardial infarction. It occurs, but is infrequent in cases of mitral stenosis, thyrotoxicosis, and other conditions which lead to failure of the left ventricle. Chronic pulmonary diseases, particularly chronic bronchitis, emphysema, bronchiectasis tuberculosis, and carcinoma have been said to predispose to cardiac asthma, but on very little evidence.

The mechanism by which paroxysmal cardiac dyspnea is generated, with or without asthma, can be described as follows: When the predisposed person falls asleep, the respiratory reflexes become less sensitive, ventilation is adequate, and there is functional balance between the right and left ventricles. The sequence of events leading to an attack of cardiac asthma begins soon thereafter. This sequence should be thought of as progressing through three stages, namely, the events which precipitate, those which maintain, and those which relieve the attack.

Factors which tend to precipitate an attack are thought to have in common the ability to change the cardiopulmonary status from that of the depression of sleep to the reflex hyperexcitability of the awakening. This, in

turn, is thought to lead to increased ventilation, pulmonary congestion, reduced vital and maximal breathing capacities, and a temporary imbalance between the right and left ventricles. The things to which this sequence of events have been attributed are: the supine position, resorption of edema fluid, increase of venous pressure, a decrease in the concentration of the serum protein, a decrease in vital capacity during the early morning hours, an accumulation of bronchial mucus, deep breathing, cough, nightmares, abdominal distention, constipation, a full bladder, hunger, excessive warmth, arrhythmia, outside noises, Cheyne-Stokes respiration, trepopnea, nausea and vomiting, decreased coronary flow during sleep in cases of hypertension, muscular effort of an attack of noncardiac asthma, and compression of the pulmonary veins by the enlarged left ventricle.

Factors which tend to maintain an attack are said to be the persistence of those which precipitated it and the added metabolic demands of the cough and strenuous respiratory efforts of the attack. The vicious circle of cough—increased ventilatory effort, pulmonary congestion, and cough—leads to pulmonary edema or to acute coronary insufficiency and death unless it is broken.

Factors which tend to relieve an attack have been reported to be expectoration of mucus, emptying of the bladder, passage of gas, throwing off the bed clothes, and assumption of the upright position. If these factors do not produce relief promptly, the physician can relieve the attack by depressing the respiratory reflexes and cough, by decreasing the return of venous blood from the periphery, by decreasing peripheral vascular resistance, by reducing bronchospasm and anoxemia, by increasing the flow of blood in the coronary vessels, and finally, by improving myocardial efficiency.

Fortunately, most attacks can be relieved by the administration of morphine alone or supplemented by the application of tourniquets to the four extremities (bloodless phlebotomy). The efficiency of drugs and other measures used for the relief of asthma depends on their ability to increase the coronary circulation, to decrease the volume of circulating blood, peripheral vascular resistance, hyperventilation, and bronchospasm, and to restore the normal function of the left ventricle of the heart.

Administration of small doses of a 1 : 1000 solution of epinephrine is effective in the treatment of cardiac and noncardiac asthma. The sympathetic nerves are thought to be coronary dilators. Administration of small doses of epinephrine relaxes the bronchioles and arterioles, increases the flow of blood in the coronary vessels, and increases the functional capacity of the left ventricle.

The role of the allergist has been reemphasized recently in a review of 26 cases of cardiac asthma. Several clinical observations were made which are thought to have practical value. For example, asthma and heart disease may run their courses independently, neither seeming to influence the severity of the other. Typical cardiac asthma and typical nonasthmatic

paroxysmal cardiac dyspnea can occur at different times in the same patient. An attack of asthma due to other causes may occur in a case of cardiac disease in which there is no evidence of heart failure; heart failure may occur in a case of asthma in which there is no intensification of pre-existing asthma. Mild attacks of cardiac asthma may precede a series of severe attacks. A series of mild nightly attacks of cardiac asthma is not infrequent. Cardiac asthma may be relieved temporarily by noncardiac management including the use of conventional symptomatic remedies for asthma. Patients with severe heart disease may often avoid attacks by avoiding known allergenic influences. Prompt recognition of the allergic or infectious component may provide early protection of the heart from the strain of repeated attacks.

In only 6 of the 26 cases of cardiac asthma studied with the disciplines of the allergist and the cardiologist were the patients relieved adequately by cardiac therapy alone. The other patients required cardiac plus allergy and infection therapy for maximal relief. Eighteen had had asthma due to other causes. Twenty-five—all but one—had other manifestations of an allergic background. Fifteen had evidence of respiratory infection. All had some evidence of heart failure, usually from arteriosclerotic heart disease, with or without hypertension.

The treatment of cardiac asthma is rather routine after the role of paroxysmal failure of the left ventricle has been recognized and the other noncardiac causes of asthma have been evaluated. Attacks of asthma and the underlying heart failure are controlled by the measures described. Treatment of allergy consists of the avoidance of offending foods and inhalants and the injection of extracts of allergens which cannot be avoided. Infection should be treated promptly and persistently until cured by specific drugs and other appropriate therapy. The paranasal sinuses should receive particular attention. (Swineford, O. Jr., Cardiac Asthma: Postgrad. Med., 24: 577-584, December 1958)

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Acute Venous Thrombosis

The urgency of the physician's concern with acute peripheral venous thrombosis (phlebitis) clearly lies in the intimate but unpredictable association between clots in peripheral veins and death from embolization of these clots to pulmonary arteries. This article reevaluates the problem of phlebitis on the basis of critical review of published data and of clinical, pathologic and experimental observations. It is believed that a useful frame of reference can be derived from this presentation whereby a rational choice of therapy can be made in a large majority of clinical situations.

When thrombosis of a peripheral vein is accompanied by local venous distention, tenderness, edema, discoloration, pain, and fever, the diagnosis

is not difficult. Similarly, phlegmasia alba dolens presents little diagnostic challenge and phlegmasia cerulia dolens—a form of massive venous thrombosis that may occasionally be mistaken for acute arterial occlusion—is usually easily recognized. However, less extensive venous thrombosis may present extraordinary diagnostic difficulties to the most experienced clinician. Calf tenderness, pain on dorsiflexion of the foot, slight elevation of the postoperative pulse and temperature, and minimal edema of the extremity are described by most authorities as the early evidences of deep venous thrombosis. Yet, anyone who has seen patients with such symptoms knows how difficult they may be to evaluate. Dubious calf tenderness, which may be the only clinical expression of a potentially serious venous obstruction, is even less diagnostic and may be due to orthopedic defects, arthritis, myxedema, or myositis.

Various physical signs or maneuvers have been recommended as an aid in the diagnosis of phlebitis. None of these has as yet either stood the test of time or been widely accepted. Laboratory tests have been similarly unrewarding. Venography which enjoyed a brief period of popularity has fallen from general use. The heparin tolerance—although employed in some clinics—has not gained wide acceptance. Several investigators have described alterations in known clotting factors in disorders frequently associated with thromboembolic phenomena, but correlations between biochemical abnormalities and clinical phlebitis have not been forthcoming. All these direct and indirect attempts at diagnosis have failed because there is still no reliable test for detecting the prethrombotic, incipiently thrombotic, or actively thrombotic state. Furthermore, the severity of the phlebitic symptoms cannot readily be correlated with the likelihood of symptomatic or lethal embolization.

Massive venous thrombosis is often not attended by recognizable pulmonary embolism, whereas minimal clinical evidence of a clot in a peripheral vein can be associated with a fatal obstruction in a pulmonary artery. This observation has led to a distinction between "phlebothrombosis" and "thrombophlebitis." In the former, the clot is believed to be the predominant aspect of the pathologic process and inflammation of the vein wall a secondary phenomenon; in the latter, the reverse is believed to obtain. It has been stated that the hazard of pulmonary embolism is greater in phlebothrombosis. Actually, the edema, cyanosis, pain, and fever that accompany some episodes of phlebitis may be related more to the extent of the venous obstruction than to actual inflammatory changes in the vein wall. In any event, this classification appears to be of dubious prognostic value; it may on occasion give rise to a false sense of security.

Few physicians would deny that venous thrombosis—difficult as it may be to diagnose on occasion—is also a common and usually benign disease. Such a clinical constellation obviously creates many obstacles to defining a practical and consistent therapeutic program. On the other

hand, the unexpected fatality, occasionally in otherwise healthy individuals, strengthens the physician's desire to apply a therapeutic program to all patients suspected of having venous thrombosis.

There are three additional facets to the pathogenesis of venous thrombosis that have not received adequate recognition, but that nevertheless influence the course of the disease to a profound degree. The first is that venous thrombosis has a definite, if indeterminate, incidence of recurrence. When a patient is first seen, it is not possible to predict in that individual whether a recrudescence may appear, where it may become clinically apparent, or how extensive and enduring it may prove to be.

Secondly, venous thrombosis is frequently a focal manifestation of a systemic disease. Evidence to support the systemic nature of venous thrombosis derives from patients who have multiple episodes, or phlebitis in different areas of the body with or without underlying recognizable systemic disease. Recurrence of migratory superficial and deep phlebitis in the extremities, venous thrombi in visceral and cerebral veins, right atrial thrombi, and nonbacterial thrombotic endocarditis are examples.

Thirdly, the extent to which intravascular thrombi may dissolve spontaneously has not been adequately appreciated. Clinical observations of the natural history of venous thrombosis in superficial veins in man clearly indicate that obstructed vessels may in time become completely patent. Fibrinolytic activity in normal blood has been measured in vitro and spontaneous increases in lytic activity have been observed in a variety of pathologic states.

All of these considerations relating to the diagnosis and clinical course of venous thrombosis indicate the extreme difficulty—in the present state of knowledge—of a statistical approach to the clinical evaluation of therapy for phlebitis and may, in fact, account for the varying conclusions reached by different clinical investigators.

To construct a workable frame of reference for the clinical management of venous thrombosis within the limits of present knowledge, the authors have formulated a general concept that has been helpful to them in the treatment of phlebitis. This concept is predicted on the assumed existence of a hypercoagulable state and hypothesizes that local vascular injury or stasis alone is inadequate to induce clinically significant venous thrombosis. The hypothesis states that individuals with clinically recognized intravascular clotting have a state of hypercoagulability as yet not demonstrable that predisposes them to thrombosis. Hypercoagulability may be due either to known or as yet unidentified alterations in one or more specific clotting constituents. These alterations may be induced by the liberation into the circulation of altered moieties from gravid uteri, traumatized tissues, necrotic foci, malignant neoplasms, or by systemic changes in plasma proteins. When the balance between clot formation and clot lysis is altered, a thrombus can be initiated and potentiated by a degree of stasis that would not

by itself result in clot formation. Finally, hypercoagulability and vascular stasis may be systemic or local, transient, prolonged, or recurrent. With this formulation which they have termed "the temporary thrombotic state" treatment becomes predicted upon neutralization of the hypercoagulability by retarding the deposition of further clot (anticoagulants); the dissolution of already formed fibrin (lytic agents); and upon the reduction of stasis through the cessation of procedures that favor immobilization and the correction of factors, such as shock, congestive failure, and a large peripheral venous pool that favor retarded blood flow.

Because the status of lytic agents is still experimental and the correction of shock and congestive failure are well established on other grounds, the specific treatment of acute phlebitis is reduced at present to the neutralization of hypercoagulability by anticoagulant drugs and the decrease in vascular stasis by reduction of immobilization and the use of elastic supports. Within this formulation, vein ligation and other measures proposed for the relief and control of venous thrombosis become ancillary therapeutic tools.

This review outlines the basis for proposing, and the details for implementing, a rational and consistent plan of therapy for the management of acute venous thrombosis. The authors interpret the entity of venous thrombosis as representing a common, frequently systemic disease, clinically manifest by focal intravascular coagulation. Concerning this disease, the specific etiology is unknown, the diagnostic criteria are crude and unsatisfactory and, although the clinical course is usually benign and self-limited, it may be unpredictably recurrent and fatal. They have also inferred that the difficulties in statistically validating any specific therapeutic measure are, for the present, extremely great and that this problem will not be significantly lessened until an accurate test for thrombosis is devised.

To construct a workable frame of reference for the clinical management of venous thrombosis within the limits of present knowledge, the authors have formulated the concept of the temporary thrombotic state. Treatment is based upon the neutralization of hypercoagulability by anticogulant drugs and upon a reduction of venous stasis by the removal of immobilization and a decrease in the size of the peripheral venous pool by elastic support. Various aspects of this therapeutic approach are described in detail and illustrated by selected case studies. (Wessler, S., Deykin, D., Theory and Practice in Acute Venous Thrombosis - A Reappraisal: Circulation, XVIII: 1190-1207, December 1958)

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Use of funds for printing this publication has been approved by the Director of the Bureau of the Budget 19 June 1958.

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Benign Diseases of the Uterine Fundus

Benign disorders of the uterine fundus, exclusive of infections, make up a large part of gynecologic practice and frequently are both misdiagnosed and mismanaged. A review of the most common of these conditions should serve to clarify their diagnosis and outline their management.

Mucosal Hyperplasia

Various synonyms have been applied to this condition in the past. The most common ones are glandular hypertrophic endometritis, hemorrhagic metropathy, and glandular hypertrophy of the endometrium. A preferred term is hyperplasia of the endometrium or mucosal hyperplasia of the uterus.

Treatment of endometrial hyperplasia depends largely on the age of the patient, intensity of bleeding, and degree of anemia. In young women and girls, administration of progesterone in a dosage of 50 to 100 mg. per day for 3 days at 4-week intervals will decrease the bleeding and may solve the problem for an extended period. If bleeding is excessive, androgenic therapy may be offered to suppress bleeding in a short time. Testosterone propionate 50 mg. per day for 4 days, or methyltestosterone, 20 mg. for 5 days may serve this purpose without the necessity for hospitalization and dilatation and curettage. If progesterone and androgen fail to control the bleeding or if it is grossly excessive requiring transfusions, dilatation and curettage are indicated.

In middle-aged or menopausal women, the incidence of malignant disease of the endometrial cavity is higher and for this reason, one should perform dilatation and curettage earlier in the course of the disorder. After diagnostic procedures, such as cytologic examination, endometrial biopsy, and hysterography have been carried out, one should first perform dilatation and curettage for diagnostic purposes and then await its therapeutic effects. Use of progesterone as outlined or testosterone, or both, frequently results in regression of symptoms and control of the problem. Some workers have suggested radiation of the hypophysis as an approach to endometrial hyperplasia, but it is not widely used. Partial wedge resection of the ovaries has proved useful, particularly in the presence of the Stein-Leventhal syndrome. Amenorrhea is more common in the latter condition than excessive menstrual flow.

Adenomyosis (Endometriosis Interna)

Synonyms for adenomyosis have included direct endometriosis, adenomyoma, adenomyositis, endometrioma, adenomyohyperplasia, hysteroadenosis, fibroadenomatosis, and adenoma endometrioides. The most widely accepted term at present is adenomyosis, the definition of which is simply ectopic intramural uterine endometrium.

Hysterectomy is required when the patient has signs and symptoms of a degree sufficient to warrant therapy. Hormonal therapy and radiation therapy are rather poor second choices.

Myometrial Hyperplasia (Fibrosis Uteri)

Terms used in the literature to describe this disorder include chronic metritis, diffuse myoma, fibrosis uteri, and congestion fibrosis. The acceptability of these terms is currently under dispute and discussion is somewhat limited.

Treatment of myometrial hyperplasia is hysterectomy because it does not respond to endocrine control. The menorrhagia that accompanies the disorder may be sufficiently grave to warrant surgical intervention.

Endometrial Polyp

Endometrial polyps are frequently associated with endometrial hyperplasia. In the past, they have been called adenoma polyposum. The presence of a polyp in the postmenopausal or menopausal period suggests excessive stimulation of the endometrial lining and a response of the endometrium to the stimulus of neoplastic quality. There is a growing feeling that endometrial polyps may have the same role in the development of endometrial adenocarcinoma as colonic polyps have in the development of adenocarcinoma of the colon. Although the incidence of malignancy in association with endometrial polyps is appreciably lower than that of carcinoma in association with colonic polyps, one should, nevertheless, keep the relationship in mind in managing the condition. Certainly, it suggests that treatment might well be more radical than simple curettage and polypectomy; hysterectomy is being offered more and more when contraindications to it are not present.

Myomas and Fibromyomas

Myomas and fibromyomas have been defined as benign discrete nodular tumors of the uterine wall composed of uterine muscle and fibrous tissue in varying proportions and arranged in a disorderly manner. They have also been called fibroids and leiomyomas.

Regular follow-up observation at intervals of 4 to 6 months should suffice if uterine fibroids are asymptomatic, small in size, and unrelated to infertility. The majority of fibroids may never require therapy under these conditions. If the fibroid is moderately large and associated with sensations of weight and pressure, pain or disturbed uterine bleeding, multiple myomectomy is a procedure which has long been recommended for younger women. It is being more widely applied by gynecologic surgeons. Certainly, in the presence of infertility, it is highly desirable to remove the fibroids and preserve the uterus. Postmenopausally, it is best to perform hysterectomy for fibroids, particularly if they are the size of a 4-month gestation or larger or associated with sensations of weight, pressure, and pain, and signs of necrosis or bleeding.

Lipoma, Osteoma and Chondroma, Hemangioma and Lymphoma

Various benign desmoid tumors may develop in the uterus independently;

when associated with uterine sarcomas they are designated as teratoid tumors. The great variety and the many minor differences in growth and invasive capacity of the mixed tumors make classification difficult. The diagnosis is almost always made by the pathologist because the majority of these tumors are called "fibroids" preoperatively. All are usually treated by hysterectomy. (Kasdon, S. C., Benign Diseases of the Uterine Fundus: Postgrad. Med., 24: 464-474, November 1958)

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Patella in Degenerative Joint Disease

A detailed description of the patella in degenerative joint disease in a large series of cases has not been reported in the literature. The marked increase in the number of patellectomies for this disease in recent years has made this study possible.

The patella is a triangular bone, the apex of which points distally; this shape is best recognized from the anterior or non-articular surface. The posterior surface is divided into an oval articular region above, and a non-articular apex below to which the infrapatellar fat pad is attached.

During the 5-year period, 1950 through 1954, a total of 154 patellectomies were carried out on 136 patients with degenerative disease of the patellofemoral joint. There were 85 female and 51 male patients. All patients presented with the complaint of pain. Associated complaints were locking, instability, stiffness, crepitus, and swelling. Physical findings noted were tenderness, crepitus, effusion, and limitation of motion. The histories could be divided into two groups depending on the presence or absence of a predisposing condition. An etiologic factor was present in 89 cases while in the remaining 47 cases, no predisposing condition was found.

There was a history of trauma in 45 cases. In 13 cases, there was an old fracture of the patella, the lower end of the femur, or the upper end of the tibia. A torn semilunar cartilage was found at operation in 9 patients. The remaining 23 patients gave histories of injury, but specific findings were not observed.

A history of recurrent dislocation of the patella was given by 20 patients, of whom 18 were female and 2 were male. In 9 cases, dislocation was bilateral; all 9 patients were female. These patients complained of pain and instability or dislocation. The youngest patient was 15 years of age and the oldest was 58 years of age; the average age was 35.

The patellas were available in 109 cases; in 10 patients, both patellas were removed, making a total of 119 patellas examined. Each patella was anatomically oriented and inspected for the presence of cartilage, degeneration, eburnation of the articular surface, and marginal hyperplasia. The bone was then sectioned horizontally through the center so that a cross section

of the vertical ridge was obtained. The patellas were divided into three grades by means of the following criteria: Grade 1, cartilage degeneration with absence of, or slight, marginal hyperplasia; Grade 2, cartilage degeneration with well marked marginal hyperplasia; Grade 3, gross distortion in the shape of the patella with eburnation of the articular surface. This method of grading is simple and represents the progression of degenerative changes.

Degenerative disease of the patello-femoral joint is commoner among women than among men. In seeking an explanation for this, the known etiologic factors must be examined. The three most frequent conditions predisposing to degenerative disease of the patellofemoral joint are trauma, obesity, and recurrent lateral dislocation of the patella.

Trauma is commoner among men; this explains the higher incidence of male patients having degeneration of Grades 1 and 2 than degeneration of Grade 3. The relative absence of trauma among patients having Grade 3 degeneration probably reflects earlier surgical intervention in cases in which trauma was a predisposing factor. It is interesting to note that the incidence of overweight of 30 pounds or more was approximately the same for men (7 of 30, or 23%) as for women (15 of 58, or 26%) with degenerative disease of the patellofemoral joint because this condition is generally thought to be commoner among women.

Recurrent lateral dislocation of the patella is commoner among women and it has been shown that dysplasia of the lateral margin of the trochlea and of the patella itself is frequently present in these cases. Lateral subluxation of the patella frequently occurs in advanced degenerative joint disease and is commoner among women.

Patellectomy for degenerative joint disease gives subjective improvement in a high proportion of patients, particularly those with advanced disease. (Haliburton, R.A., Sullivan C.R., The Patella in Degenerative Joint Disease: Arch. Surg., 77: 677-682, November 1958)

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Broncho-Pulmonary Amoebiasis

Amoebiasis is mainly and primarily an infection of the colon by the pathogenic amoeba, Endamoeba histolytica, but it is also characterized by a variable clinical course in which the manifestations caused by the occurrence of complications may dominate the picture.

Invasion of the submucosa may be followed by entry of E. histolytica into the radicles of the portal vein and metastasis of the infection to the liver. This is followed by amoebic hepatitis or amoebic abscess of the liver which is by far the most frequent and important complication. However, it has always been known that the lung could be involved and that if

the physician—particularly in an endemic area—is amoebiasis conscious, he will be able to find out that amoebic infection is the real cause of many cases that would otherwise have been diagnosed as pneumonitis or lung abscess of undetermined or unknown etiology.

Possible routes of infection are:

1. Primary pulmonary amoebiasis: where endamoeba reach the lung by direct embolism from the bowel through the middle and inferior hemorrhoidal veins. In these cases, pulmonary amoebiasis may develop independently of hepatic affection and moreover the pulmonary condition does not necessarily manifest as suppuration, but it may simulate bronchopneumonia or miliary tuberculosis. Autopsy of such cases revealed firm consolidated nodules in the lung in which endamoeba were demonstrated in the section.

2. Secondary pulmonary amoebiasis: This is the commonest variety. The condition is associated with, or can be related to, an amoebic liver disease. In such cases, infection spreads from the intestines to the liver through the portal circulation and then reaches the lung: (a) by direct extension through the diaphragm; or (b) by embolization from thrombosed hepatic veins to the inferior vena cava to the right side of the heart and through the pulmonary arteries to the lungs.

Recently, cases of lung suppuration have been reported, caused by endamoeba gingivalis, a non-pathologic saprophyte which may be aspirated into the bronchial tree and produce disease.

In primary cases, amoeba after reaching the lung produce focal pneumonic consolidation soon followed by necrosis and softening as in the liver. With the discharge of the necrotic tissue into the pleural space or through the bronchi, a cavity is formed and the lobe undergoes, in part or all, a process of consolidation.

Some have thought that suppuration may commence between the diaphragm and the liver or in the base of the lung, but extensive Calcutta experience as reported by Roger shows that this is rarely, if ever, the case. What happens is that the capsule of the liver becomes fused with the diaphragm and the pus escapes into the surrounding tissues to such an extent that the primary liver abscess cavity may shrink to the size of the tip of the finger and escape observation while a small opening leads into a large secondary abscess in the base of the lung or between the liver and the diaphragm.

In most cases, diagnosis was based on evidences obtained from the history, the right basal localization of the lung lesions, the character of the sputum, and the associated liver condition in the presence of laboratory findings that could exclude other possibilities. Diagnosis is more assured by failure of other specific remedies and the dramatic responses to emetine.

Difficulties were met in differentiating between pulmonary tuberculosis and pneumonias, including the suppurative forms. The long observation without specific treatment or under drugs other than antiamoebic ones without

improvement, compared with the rapid and dramatic improvement that progressed to complete cure once antiamoebic treatment was started, should clarify etiological diagnosis.

Emetine hydrochloride was used in all cases with remarkable success, so much so that it is justifiable in doubtful cases as a therapeutic test. The general belief is that this is a specific drug and under experimental conditions destroys E. histolytica in a dilution of 1 : million, although Dobell showed that it is lethal to amoeba in culture in a strength of 1 : 5 millions. It is reported to be more effective in metastatic lesions than in intestinal infection and Manson Bahr advises the following as a full course:

One grain daily for 7 days, then two grains daily for 4 days, and then one-half grain daily for 3 days.

(Abdel-Hakim, M., Higazi, A. M., Cairo, Egypt, Broncho-Pulmonary Amoebiasis: Dis. Chest, XXXIV: 607-617, December 1958)

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Teflon (Tetrafluoroethylene Resin)

(This article was prompted by the repeated rumors during the past 12 months regarding a machinist who reportedly died as a result of smoking a cigarette contaminated by Teflon chips. Although the rumor has appeared in several publications, a thorough investigation by the Air Force and others has proved it to be completely unsubstantiated. The Bureau of Medicine and Surgery has received no reports on toxicity from the field involving any untoward effects due to the use of Teflon.)

Teflon, because of its unusual engineering properties, has been used successfully and without health hazard in a wide variety of naval applications. This plastic combines a high degree of chemical resistance, dielectric strength, and thermal stability. As a result, valve packings, diaphragms, and electrical insulation are being fabricated from this plastic for use under circumstances where other materials could not be used.

Recently, some questions have been raised concerning the potential health hazard during operations with Teflon. The principal manufacturer of this plastic made an exhaustive survey of this subject. The following information summarizes the results of this survey and includes data obtained through 20 years of experience by the du Pont de Nemours Company, hundreds of processors and their employees, and thousands of end-item users handling this plastic daily:

1. Teflon should be regarded as an inert material. It can be eaten in food, consumed in drink, or worn close to the skin without danger. There have been no permanent injuries or deaths in the use of Teflon resins.

2. Decomposition Products at Elevated Temperatures. Some amplification may be desirable concerning the use of Teflon resins at higher temperatures. Under circumstances where other materials might well fail, a number of Teflon resins are designed to operate at temperatures over 400° F. While these Teflon items can and do retain their engineering properties indefinitely in such use, some minute proportions of decomposition products are evolved at a slow rate at 400° F. The rate of decomposition increases somewhat if the operating temperature is raised to 600° or 700° F. Routine use of mechanical ventilation is considered a sufficient precautionary measure. The following ventilation rates are recommended:

Product	Temperature	Ventilation Required
Teflon 1,5, or 7x	450° F.	7 cu ft/hr/lb of Teflon
	500° F.	30 "
	600° F.	70 "
	700° F.	600 "
Teflon 6, or 30	450° F.	30 cu ft/hr/lb of Teflon
	500° F.	80 "
	600° F.	600 "
	700° F.	4400 "

These air requirements represent the volume of air necessary to reduce the concentration of the gaseous pyrolysis product (from 1 pound of polymer) to 1 ppm by weight. The environment on long submergence time submarines presents a special case possibly requiring some adjustment of this limit and the accompanying air requirements. A separate study is being made of this case and will be reported subsequently.

3. In the few cases reported where excessive amounts of these fumes have been inhaled accidentally, they have caused temporary symptoms similar to grippe. These symptoms do not appear until 2 to 6 hours after exposure and pass off within 36 to 48 hours.

In summary, the following information may be used as a general guide for personnel handling, machining, or heating Teflon:

1. During the machining of Teflon, dust may be generated. Use local mechanical ventilation to control this dust. The use of a coolant is also recommended because it permits higher cutting or grinding rates and simultaneously keeps the dust concentration to a low level.

2. If Teflon resins are heated for extended periods in the 400° to 600° F. range, minute quantities of decomposition products are evolved.

Therefore, adequate mechanical ventilation should be provided when working with Teflon at temperatures greater than 400° F.

3. Smoking should be prohibited in areas where Teflon is being fabricated in order to minimize the possibility of contaminating the pipe or cigarette with Teflon dust.

(Indust. Toxicology Section, OccMedDispDiv, BuMed)

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Beryllium and Berylliosis

Beryllium performs heroic functions in hundreds of everyday products even though its total annual production is measured in pounds. A little beryllium goes a long way, especially in the alloys which account for most of its consumption. Beryl is the single industrially significant beryllium-bearing mineral. The small size of the beryllium ion favors its diffusion as a minor constituent in numerous minerals and discourages the formation of high-concentration beryllium minerals.

The principal uses of beryllium stem from the discovery in the 1920's that the addition of only 2% of beryllium to copper forms an alloy six times stronger than copper. Beryllium-copper alloys stand up at high temperatures, have great hardness, show resistance to corrosion, do not spark, and are nonmagnetic. They are used in the critical moving-parts of aircraft engines and in the key components of precision instruments, mechanical computers, electrical relays, switches, and camera shutters.

The ill effects on workers who handled beryllium compounds were first observed in the United States when a worker employed in processing beryllium ores died in 1940. By 1946, the hazard that threatened beryllium workers developed an alarming new aspect of the disease produced. Long after exposure had ceased, 17 cases of chronic beryllosis were reported in persons previously engaged in the manufacture of fluorescent lamps. Most of the cases reported up to that time had been acute attacks arising during exposure to beryllium compounds.

The cases of delayed or chronic illness clearly implicated beryllium itself as the poison. The principal distinction between the various beryllium compounds now appeared to be merely their relative solubility in the body fluids. The acid-forming compounds are the most soluble and produce immediate illness. The chronic form of berylliosis is distinguished from the acute form chiefly by the delay between exposure to beryllium and the onset of symptoms.

As yet, the biochemistry of beryllium poisoning is little understood. The ion of the metal, dissociated from the oxide or salt in solution with the body fluids, appears to be the active principle. It is known that the chemical reaction of beryllium ions in the body invariably involves a hydroxide

group (OH) attached to a benzene ring. Such phenolic hydroxide groupings are found in the amino acid tyrosine which in turn undergoes metabolic transformation to such compounds as adrenalin which react with beryllium ions. In the test tube, beryllium inhibits the action of many enzymes. Injections of tiny amounts of its compounds in experimental animals cause massive damage to the cells of practically every organ with which the compounds come in contact. Similarly, in human victims, upon autopsy, lesions are found scattered throughout the organs of the body as well as in the lungs. Beryllium poisoning is now accepted as a general disease and it is recognized that the patient's condition is worse than the lung picture suggests.

Examination of the lumps and nodules that form in the tissues suggests that beryllium can cause the growth and proliferation of cells. This suspicion has been supported by the discovery that beryllium may induce cancer in experimental animals. Lung and bone cancer have been induced by intravenous injection of beryllium compounds; therefore, beryllium may be a factor in the genesis of certain cases of human lung cancer.

Although little is known about the toxicity of beryllium, investigators have made one significant finding which already facilitates diagnosis and may lead to the discovery of an effective treatment. Patients invariably exhibit a kind of allergic response to beryllium compounds. It would seem, therefore, that beryllium must combine with protein in the body to form an antigen. The antigen stimulates the formation of beryllium-specific antibodies. As a result, in all cases of active berylliosis, a small amount of any beryllium compound placed on the skin produces a local allergic reaction. This "patch test" is now a valuable aid in the diagnosis of beryllium poisoning. That beryllium forms compounds with protein suggests that the logical approach to treatment is to look for a way to tie up beryllium chemically so that it can no longer react with substances in the body.

Can the beryllium hazard be controlled? Is it possible to work with beryllium compounds in safety? The answer is a somewhat qualified "Yes." The problem reduces itself to one of good industrial housekeeping which means keeping the levels of beryllium low. But how low is low? One important factor to be taken into account is that different forms of the same beryllium compound may have radically different degrees of toxicity depending upon particle size as well as solubility. On the basis of extensive tests, the Atomic Energy Commission in early 1950 concluded that all known cases of the acute disease could be attributed to air concentrations of soluble salts in excess of 100 micrograms per cubic meter, and that when the air level exceeded 1000 micrograms of beryllium, nearly everyone developed acute berylllum poisoning. To minimize the risk of the chronic disease, on the other hand, the Atomic Energy Commission found it necessary to recommend a limit of 2 micrograms per cubic meter. The strictness of this standard can be appreciated when it is realized that the corresponding limits for dusts of other metals, such as lead, mercury, arsenic, and cadmium range from

100 to 500 micrograms per cubic meter. As to the air in the neighborhood of a beryllium plant, the recommended limit is 1/100 of a microgram per cubic meter. To the best knowledge, no cases of beryllium poisoning have appeared in or around plants which have adopted these rigid standards.

The story of beryllium highlights the whole problem of occupational disease in the present era. Advances in technology now develop so rapidly that the rare material of yesterday becomes the widely used material of today. The beryllium mishaps teach the lesson that the harmlessness of a material cannot be taken for granted; a new material must be regarded as harmful until proved otherwise. Industrial medicine must provide safe working conditions before harm results; public health agencies must see that the public is not exposed to fumes from industrial processes until safe tolerances are known. (Schubert, J., Beryllium and Berylliosis: Scient. Am., 199: 27-33, August 1958)

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Cataracts and Ultra High Frequency Radiation

Radar and communication installations are making use of increasing amounts of power. As a result, military and civilian populations may be exposed to larger doses of radio frequency energy. Such exposures will be to the body as a whole. The frequencies used probably will range from the ultra-high-frequency band at a few hundred megacycles per second to the microwave band at several thousand megacylces per second.

There is no doubt that microwave radiation has produced cataracts in animals. In these experiments, intense radiation was applied locally to the eye. The consequences of local irradiation may have some practical importance in human beings because microwave radiation is used therapeutically about the eye. There is one reported case of cataracts in a human being allegedly due to microwave irradiation, but this probably cannot be accepted as proved. In this reported case, the radiation intensity reaching the eyes was difficult to estimate. Moreover, the morphologic changes in the lenses were nuclear cataracts and not what one expects with radiation.

The objective in the present study was to determine whether or not cataracts in animals might result from exposure of the whole body to radiation at frequencies similar to those used in some high-power installations. The immediate concern was with a frequency in the range of 400 megacycles. Particular interest lay in comparing the dose which produces cataracts with that which causes death.

Only one similar series of animal exposures appears to have been made with comparable frequencies in the ultra-high-frequency band. No cataracts were observed in any of these animals, although they received radiation in

near lethal amounts. However, the paucity of reported observations directed to the problem of eye damage and the practical importance of the problem warranted further study using present methods of quantitative investigation.

Material and Methods

Most of the exposures were carried out in a waveguide system, but some were made in "free space." The term "free space" must be interpreted with some reservation. In an attempt to duplicate actual exposure conditions, the test animals were contained in a small lucite box placed in the vicinity of the radiating antenna. Adult male albino rabbits were used exclusively.

Results of Eye Examination

Examination of the eyes showed nothing which could be called a cataract. Punctate opacities were occasionally seen in the posterior subcapsular cortex of the rabbit lenses, but these were often seen prior to irradiation and were as frequent in the control group as in the irradiated group. They were of such small dimensions that they might well have been seen on one examination and overlooked on the next.

Comment

Previous investigators applying local microwave irradiation to the eyes of rabbits have reported the threshold dose for cataracts to be in the range of 120-220 mw/cm.^{2}. Finding that ultra-high-frequency radiation at an intensity of 60 mw/cm.^{2} is not cataractogenic is consistent with this, but further comparison is probably unwarranted in view of the different frequencies used. However, one might expect that the cataractogenic dose would be higher with ultra-high-frequency radiation than with microwave radiation which gives rise to heating in the region of the lens.

There is no direct way of comparing the susceptibility of human and rabbit lenses to this form of irradiation. However, with local microwave irradiation the cataractogenic dose for monkeys has been found to be higher than that for rabbits. Until evidence is presented to the contrary, these results with rabbits probably provide an assessment of the cataractogenic possibilities in human beings.

Summary

Ultra-high-frequency radiation (at 468 megacycles per second) was repeatedly applied to rabbits in doses near the lethal level. This radiation was distributed over the whole body. No cataracts developed following the exposures. (Cogan, D.G., et al, Cataracts and Ultra-High-Frequency Radiation: Arch. Indust. Health, 18: 299-302, October 1958)

(OccMedDispDiv, BuMed)

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Prolonged and Recurrent Industrial Dermatitis

Contact dermatitis occurs more frequently than any other disease arising from employment. A wide variety of causes can produce occupational dermatoses, but primary irritant and sensitizing chemicals are responsible for the majority of cases. Generally, the diagnosis of a contact dermatitis can be made by carefully observing the type of lesions present and the areas of involvement. The causes can usually be found by taking a detailed history, with special attention being paid to the time of onset and the substances to which the individual has been exposed both at work and elsewhere, and by properly performing the patch test and such other tests as may be necessary. A close observation of the course of the disease is no less important. Cure or alleviation is accomplished by eliminating contact with the irritant or sensitizing substance if possible, by proper local therapy, the use of protective clothing, by job change, and sometimes by the discontinuance of work for varying periods.

Most cases of occupational dermatitis are easily managed by proper diagnosis and appropriate treatment. However, in some cases, dermatitis recurs repeatedly or fails to heal after conventional therapy. The reasons for this—in the opinion of the Committee on Occupational Dermatoses—are (1) incorrect dermatological diagnosis, (2) failure to recognize the cause, (3) failure to eliminate the cause when recognized, (4) improper treatment, (5) failure to recognize secondary infections, (6) lack of personal cleanliness by the worker because of poor cleansing facilities and cleansing agents, (7) placement of dermatitis-prone individuals on potentially hazardous jobs, (8) development of cross-sensitivity, (9) development of multiple reactivites, (10) malingering, and other such factors as active focus of infection, presence of fungus disease, and presence of constitutional skin disease. (Council of Industrial Health, The Problem of Prolonged and Recurrent Industrial Dermatitis: J. A. M. A., 168: 516-520, October 4, 1958) (OccMedDispDiv, BuMed)

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Noise - Is It a Health Problem?

The permanent hearing loss produced by extended exposure to certain occupational noises constitutes a serious health problem. The solution to this problem lies in active programs of hearing conservation. Hearing conservation is primarily a medical responsibility. The importance of the physician's recognition of his role in meeting the threat to health cannot be overemphasized.

The physician must acquaint himself with the current knowledge of the relations of hearing loss to noise exposure. Misconceptions about the

hazardous effects of noise are widespread. Auditory effects of noise exposure and non-auditory effects are frequently related without justification. For example, the fact that a noise produces annoyance does not—contrary to popular belief—mean that exposure to that noise will necessarily produce a hearing loss. The annoyance caused by noise is a highly individual phenomenon and, as such, is not easily measured or predicted. The fact that a noise produces annoyance does not mean that it is bad for health.

The total amount of hearing loss produced by noise exposure depends on more variables than was originally thought. Before the diagnosis of noise-induced hearing loss can be made, the physician must have adequate information about (1) the over all sound pressure level of the noise, (2) the way the energy in the noise is distributed among the various frequencies present, (3) the typical pattern of daily exposure to the noise, and (4) the years of expected work life in the noise. Answers given by the patient cannot be assumed to be reliable and the physician must make the necessary effort to obtain the pertinent information from reliable sources. (Glorig, A. Jr., Summerfield, A., PhD., Noise - Is It a Health Problem? J.A.M.A., 168: 370-376, September 27, 1958) (OccMedDispDiv, BuMed)

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New Convening Dates for Pest Control Course

New convening dates have been announced for the basic course in Disease Vector and Economic Pest Prevention and Control offered at the Disease Vector Control Center, NAS, Jacksonville, Fla. The new schedule extends the course into a basic four-week course and offers the course every other month instead of quarterly.

Convening Dates for Calendar Year 1959

2 March 1959	to	28 March 1959
4 May 1959	to	30 May 1959
6 July 1959	to	1 August 1959
7 September 1959	to	3 October 1959
2 November 1959	to	29 November 1959

The course is open to all active duty officer and enlisted personnel. Military civilian employees engaged in pest control activities are also eligible for this course and are urged to attend. Billeting and messing facilities are available at NAS, Jax., for both military and civilian personnel attending course. Attendance quotas are allocated and may be requested by communicating directly with the Officer in Charge, Disease Vector Control Center, Jacksonville, Fla.

Aviation Seminar

The Fourth Annual Research Reserve Seminar in Aviation Medicine will be held at the U. S. Naval School of Aviation Medicine in Pensacola, Fla., for a two-week period commencing 9 March 1959. This seminar sponsored by the Office of Naval Research in conjunction with the Naval Air Training Command and the School of Aviation Medicine, is open to Reservists of all services who have an interest in scientific and operational problems of Naval Aviation and its role in the conducting of modern warfare. Application for attendance at this seminar should be made through regular service channels. (ONR)

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Board Certifications

American Board of Dermatology and Syphilology
LTJG Julius L. Danto MC USNR (Inactive)
CAPT Clyde W. Norman MC USN

American Board of Ophthalmology
LT William F. Hoyt MC USNR (Inactive)
CDR Roger Stevenson MC USN

American Board of Otolaryngology
LT James M. Anthony MC USNR (Inactive)

American Board of Pediatrics
LTJG Alexander S. Fitzhugh, Jr. MC USNR (Inactive)

American Board of Surgery
LT John H. Dawson, Jr. MC USNR (Inactive)
CDR Donald W. Robinson MC USN
LT Shirley E. Townsend MC USNR (Inactive)

American Board of Surgery & Thoracic Surgery
LTJG Josiah Fuller MC USNR (Inactive)

American Board of Urology
LT Robert T. Braman MC USNR (Inactive)

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From the Note Book

1. At the 1958 Meeting of the New York Academy of Sciences, the title of Fellow of the New York Academy of Sciences was conferred upon Dr. Howard T. Karsner, Medical Research Advisor to the Surgeon General. Election to Fellowship in the Academy is a signal, distinguished honor, conferred upon a limited number of Members who, in the estimation of the Council, have done outstanding work toward the advancement of Science. (TIO, BuMed)

2. CAPT C. J. Gell MC USN was presented the Melbourne W. Boynton Award for Space Medicine at the American Astronautical Society's Annual Meeting. (TIO, BuMed)

3. CAPT C. E. Wilbur MC USN and CAPT M. W. Ballenger MC USN have been elected Chairman and Secretary, respectively, of the Joint Committee on Aviation Pathology for 1959. The Committee, which includes medical officers of the military services of the United States, the United Kingdom, and Canada, held its fifth business meeting at the Armed Forces Institute of Pathology on December 11, 1958. (TIO, BuMed)

4. CAPT L. J. Pope MC USN has assumed command of the U. S. Naval Medical School, NNMC, Bethesda, Md., relieving RADM C. B. Galloway MC USN who reported to the Bureau of Medicine and Surgery as Assistant Chief of Research and Military Medical Specialties. (NNMC)

5. Two cases of pericarditis due to tularemia are reported. Tularemic pericarditis may occur as the only apparent complication of tularemia, a fact of importance in diagnosing certain cases of this serious illness. If treated with streptomycin, tetracycline derivatives, or chloramphenicol, the prognosis is similar to benign sero-fibrinous pericarditis. (Dis. Chest, December 1958; C. W. Adams, M. D.)

6. Eight cases of Ehlers-Danlos' syndrome (hyper-elasticity and fragility of the skin combined with hyperlaxity of the joints) occurring in the same family are reported. The pedigree suggests a dominant mechanism of heredity of this disease. (Arch. Dermat., December 1958; K. O. Husebye, M. D., K. Getz, M. D.)

7. The ultimate place of amphotericin B in the treatment of the deep mycoses must await both a longer observation of treated patients and comparison with more recently developed therapeutic agents. It is evident that this antifungal antibiotic is highly effective initially against North American blastomycosis, histoplasmosis, cryptococcosis, and sporotrichosis when given intravenously.

In general, it is a useful agent against most infections with yeast-like fungi. (Arch. Int. Med., December 1958; J.H. Seabury, M.D., H.E. Dascomb, M.D.)

8. The dashboard femoral fracture is an injury unique to high-speed automobile accidents. Thirty fractures of this type are reported, analyzed, and evaluated as to end result. The fracture conformation is characteristic and the etiological forces are constant. Great care must be exercised so that hip and knee injuries will not be overlooked. (J. Bone & Joint Surg., December 1958; COL S. J. Ritchey MC USA, et al.)

9. The clinical and hemodynamic findings in 30 patients with congenital aortic stenosis are summarized and the potentially serious nature of this defect is emphasized. The use of left heart catheterization in the precise assessment of the severity of the obstruction and in the detection of subvalvular stenosis is described. (Circulation, December 1958; A.G. Morrow, M.D., E.H. Sharp, M.D., E. Braunwald, M.D.)

10. A method of processing arterial segments by the freeze-drying technique is presented with a description of the equipment and the steps involved. (Arch. Surg., December 1958; J.M. Erskine, M.D.)

11. This article reports the authors' experience in scleroderma correlating the electrocardiograms with the clinical, physiopathologic, and morphologic findings. (Am. Heart J., December 1958; J. Escudero, M.D., E. McDevitt, M.D.)

12. The evaluation and management of some minor injuries of the eye are discussed in Postgrad. Med., December 1958; K. L. Roper)

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IN MEMORIAM

CAPT James P. Bowles MC USN (Ret)	24 December 1958
CAPT Louis E. Tebow MC USN	29 October 1958
CAPT Henry C. Weber MC USN (Ret)	30 October 1958
CDR Russell J. Trout MC USN (Ret)	5 November 1958
LCDR LaSalle H. James DC USN (Ret)	23 November 1958
LCDR Frederick R. Haselton Sr. MC USN (Ret)	24 October 1958
LT Frank L. Titsworth MSC USN (Ret)	4 November 1958
LTJG Edward H. Meeteer MSC USN (Ret)	11 November 1958
LTJG Paul E. Robinson Sr. MSC USN (Ret)	29 November 1958
CWO Henry D. Mullins MSC USN (Ret)	23 September 1958

Recent Research Reports

Naval Dental Research Facility, NTC, Bainbridge, Md.

1. Survey of Dental Health of the Naval Recruit. IX. Relation of Father's Occupation. NM 75 01 26.04, 24 October 1958.
2. Survey of Dental Health of the Naval Recruit. X. Relation of the Father's Income. NM 75 01 26.04, 30 October 1958.

Naval Medical Research Institute, NNMC, Bethesda, Md.

1. The Influence of Dietary Inorganic Sulfate on Growth and Dental Caries in Rats. NM 75 01 00.01.04, 15 July 1958.
2. Studies on Mineral Metabolism in the Rat. II. Effect of Casein Level of the Diet on the Formation of Calcium Citrate Urinary Calculi. NM 75 01 00 .01.05, 15 July 1958.
3. Host Influences on Some Haemosporidian Parasites. Lecture and Review Series. No. 58-5, 15 July 1958.
4. Further Studies of Drug-Resistant Strains of Rickettsia Prowazeki. NM 25 05 00.02.02, 21 July 1958.
5. Studies on the Contractile Proteins of Muscle. II. Polymerization Reactions in the Myosin B. System. NM 01 01 00.02.06, 4 August 1958.
6. Radiomimetic Effect of Triethylenemelamine on Reproduction in the Male Rat. NM 01 02 00.03.02, 5 August 1958.
7. Some Changes in the Biological Characteristics of Colonized Anopheles Quadrimaculatus Say. NM 52 07 00.01.02, 6 August 1958.
8. Toxicological Study of Hydraulic Fluids Cellulube 550A, Kolube 220, and Houghto-Safe 1055. NM 53 01 00.03.01, 8 August 1958.
9. Histological Evidence of the Mechanism of Resistance to Challenging Cercariae of Schistosoma Mansoni. Lecture and Review Series No. 58-6, 8 August 1958.
10. Normal Rat Serum as a Growth Factor for Trypanosoma Lewisi. Lecture and Review Series No. 58-7, 8 August 1958.
11. Polarographic Observations on Bivalent Metallic Ion-Acetylcholine-sterase Interaction. NM 02 02 00.01.09, 14 August 1958.
12. An Experimental Study of Ammonium Intoxication. NM 72 02 00.01.02, 22 August 1958.
13. Electro-Encephalographic Correlates of Ammonium Carbonate Intoxication in the Rat. NM 72 02 00.01.03, 9 September 1958.

Naval Medical Research Unit No. 3, Cairo, Egypt

1. Ticks (Ixodoidea, Ixodidae) Parasitizing Lower Primates in Africa, Zanzibar, and Madagascar. NM 52 08 03.3.03, January 1958.

2. Notes on African Haemaphysalis Ticks. IV. Description of Egyptian Populations of the Yellow Dog-Tick, H. Leachii Leachii (Audouin, 1827) (Ixodoidea, Ixodidae) NM 52 08 03.3.02, January 1958.
3. Needle Biopsy of the Lung. NM 72 04 03.1, March 1958.
4. The Hares of Egypt. NM 52 08 03.7.02, April 1958.
5. On Zone Electrophoresis of Human Parotic Saliva in Starch Gels. NM 75 02 03.2(1), April 1958.
6. The Ticks (Ixodoidea) of Iraq: Keys, Hosts, and Distribution. NM 52 08 03.3.04, May 1958.
7. The Ticks (Ixodoidea) of Egypt. NM 52 08 03.3.07, May 1958.
8. Histopathological Study of Liver Biopsies in Pulmonary Tuberculosis in Egypt. NM 52 13 03.1.01, November 1958.

Naval Medical Research Laboratory, Submarine Base, New London, Conn.

1. The Measurement of Dextrose in Standard Solutions with Dreywood's Anthrone Reagent and the Klett-Summerson Photoelectric Colorimeter. (A statistical evaluation and simple microprocedure). Report No. 294, NM 24 01 20.02.01, 10 April 1958.
2. Memorandum Report No. 58-9. Some trends in the Submariner Selection Data for 1957-1958. NM23 02 20.01.01, 30 July 1958.
3. Memorandum Report No. 58-8. Report on a Direct-Current High-Pressure Xenon Arc. NM 22 01 20.01.03, 6 November 1958.

Naval School of Aviation Medicine, NAS, Pensacola, Fla.

1. Vocal Pitch Changes: Effects on Intelligibility Test Scores. Report No. 76, Subtask No. 1, NM 18 02 99, 30 April 1958.
2. Effect of Restricting Information in the Verbal Conditioning Situation on Extinction. Report No. 1. NM 14 02 11-11, 1 May 1958.
3. A Note on Peer Nominations as a Predictor of Success in Naval Flight Training. Report No. 14, Subtask No. 1, NM 16 01 11, 23 May 1958.
4. Calculation of Product-Moment Correlations Matrices with the IBM 604 Calculating Punch. Report No. 26, Subtask No. 1, NM 14 02 11, 1 July 1958.
5. Evaluation of Certain Visual and Related Tests. IV. Size Constancy. Report No. 4, Subtask No. 6, NM 14 01 11, 15 July 1958.
6. Spontaneous Pneumothorax in Flight - A Case Report and Brief Review of the Literature. Report No. 3, Subtask No. 1, NM 19 01 11, 17 July 1958.
7. Reflex Ocular Torsion in Healthy Males. Report No. 47, Subtask No. 1, NM 17 01 11, 30 July 1958.

(Listing of Research Reports will be continued in an early issue)

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DENTAL SECTION

Dental Corps Featured in January Journal

The January issue of tne newly named Journal of Oral Surgery, Anesthesia, and Hospital Dental Service departed from its long established publication procedure by devoting the entire issue to articles by authors who are associated with a single source. The nine authors of seven scientific articles and seven authors of five case reports are all officers of the Dental Corps of the U. S. Navy. It is anticipated, therefore, that the January 1959 issue will be recorded as the United States Navy issue. Although none of the thirteen reports deal with conditions specific to the Naval Service, that fact makes them more valuable to dentists in civilian practice because the conditions described are common to both civilian and military patients.

The titles of the editorial, articles, and case reports and the names of their Navy Dental Corps authors are:

Asepsis in Minor Oral Surgical Procedures: A Reevaluation (Editorial) - Rear Admiral Ralph W. Taylor DC USN

Blue (Jadassohn-Tieche) Nevus: A Previously Unreported Intraoral Lesion - Commander Henry H. Scofield DC USN

Management of Oral Surgical Problems Complicated by Maxillary Sinus Involvement - Captain Donald E. Cooksey DC USN and Captain Robert A. Middleton DC USN

Treatment of Zygomatic Fracture-Dislocations
Captain Walter W. Crowe DC USN

Use of the Foley Catheter in Supporting Zygomatic Fractures - Captain John P. Jarabak DC USN

Use of Freeze-Dried Bone for Treatment of Nonunion after Surgical Correction of Mandibular Prognathism - Captain Raymond F. Huebsch DC USN

Cervicofacial Actinomycosis: A Postextraction Complication - Captain Theodore A. Lesney and Commander Kimble A. Traeger DC USN

Diagnosis of Oral Keratotic Lesions -
Captain Louis S. Hansen DC USN

Case Reports
Management of Subcondylar Fracture -
Commander Anthony P. Giammusso and Captain W. Basil Johnson DC USN

Mandibular Protrusion -
Captain Harold G. Green DC USN

Mixed Tumor of Floor of Mouth in a Seven-Year Old Child -
Captain Robert A. Colby and Captain Roger G. Gerry DC USN

Simultaneous Carcinoma of Upper and Lower Lip -
Captain Frederick T. Wigand DC USN

Carcinoma in a Healing Alveolus after a Dental Extraction -
Captain Wilbur N. Van Zile DC USN

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Information Required from Essayists and Clinicians

Dental officers who accept invitations to present essays or clinics before an organization or meeting are reminded of the necessity to comply with Article 6-34, Manual of the Medical Department. This directive requires the submission of pertinent information to the Bureau of Medicine and Surgery, and to Commandants of the Naval Districts involved at the earliest practicable date prior to the meeting. Timely submission of the information will afford the Bureau and District Dental officers an opportunity to give appropriate publicity to the event.

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ADA Relief Fund Reaches $63, 695

Contributions to the ADA Relief Fund reached $63,695 on November 30, 1958, as reported by Dr. H. R. Bleier of Milwaukee, Chairman of the ADA Council on Relief. The Navy Dental Corps has contributed 51%, or $560.95, of its $1100 quota. Dr. Bleier urges that contributions be sent as soon as possible to: ADA Relief Fund, 222 East Superior St., Chicago 11, Ill. He states: "The cause is highly deserving and the need is great."

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RESERVE SECTION

Tropical Medicine in the Field
NavPers 10995 - 1958 Edition

During World War II, tropical diseases caused more casualties in many areas than did enemy action. Such diseases have long been a serious deterrent to successful military operations in the tropics. The added experience of the recent wars has naturally heightened military interest in tropical medicine and especially in the application of the many recent advances in medicine to the treatment of tropical diseases.

This correspondence course will be of special interest to all Medical Corps officers. The purpose of the course is to provide a concise guide in tropical medicine not only for the physician practicing in the tropics, but also for those in temperate zones who may be encountering tropical diseases of servicemen and others returning to the United States after a tour of duty in the tropics. The course is based upon A Manual of Tropical Medicine by Mackie, Hunter, and Worth; it covers the essential practical aspects of epidemiology, diagnosis, treatment, and prophylaxis of the more important tropical diseases.

Laboratory analysis plays an especially helpful and dramatic role in the diagnosis of diseases in indigenous personnel in tropical areas where the language barrier may make a provisional diagnosis difficult. Another factor to be considered is that natives of tropical areas may react to local diseases with symptoms very unlike those exhibited by non-natives. The text material stresses the importance of military medicine and special effort has been directed to the condensation of information essential for the Armed Forces, the clinician, the field worker, and the student of tropical medicine.

The course consists of twelve (12) objective type assignments and is evaluated at thirty-six (36) Naval Reserve promotion and/or non-disability retirement points. Naval Reserve personnel who previously completed the correspondence course, "Tropical Medicine in the Field," NavPers (none), edition 1950, will receive additional credit for the completion of course NavPers 10995.

Applications for this course should be submitted via applicant's command to the Commanding Officer, U. S. Naval Medical School, National Naval Medical Center, Bethesda 14, Md. (Attn: Correspondence Training Division).

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Some Pointers on Promotion Point Credit for Residency Training

1. Reserve Medical officers not on active duty may receive promotion point credit for completion of approved residency training.

2. One promotion point for each semester hour or equivalent thereof may be credited not to exceed 12 per fiscal year. To be creditable, residency training must have been completed in present grade since 1 July 1950. For residencies completed prior to 1 July 1957, promotion points are creditable as of 1 July 1957. Residency training completed in subsequent fiscal years may be creditable on a yearly basis as of 30 June of the fiscal year that each year's training is completed.

3. Requests for promotion point credit must be submitted by the individual Reserve officer to the Officer in Charge, Reserve Officer Recording Activity, Omaha, Neb., forwarded via the Chief, Bureau of Medicine and Surgery.

4. Each request must be accompanied by a certification from the institution or activity where the training was taken showing the type and inclusive periods in which enrolled. Credit can only be given for training completed in increments of 12 months and cannot be credited in advance of any given date.

5. The Chief, Bureau of Medicine and Surgery will evaluate the training received and by endorsement certify the number of promotion points and forward individual requests to RORA for appropriate administrative action.

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PREVENTIVE MEDICINE SECTION

DDT Resistance in the Human Body Louse

The failure of DDT to control the human body louse was first observed in Korea in 1950 - 1951. Laboratory studies conducted by H. S. Hurlbut established that these lice had a measurable degree of physiological resistance. G. W. Eddy confirmed these findings and pointed out that, although all samples of lice tested were then highly resistant to DDT, the susceptibility of Korean lice to DDT prior to the use of this insecticide was not known; that is, there was no base line for comparison in that area. Similarly,

C. H. Barnett and E. C. Knoblock reported that human body lice collected from vagrants in Tokyo were distinctly resistant to DDT and they presumed that these lice had not previously been exposed to DDT—at least for several months prior to testing.

It is impossible to ascertain from these reports whether this resistance was natural or was acquired by selection through the use of DDT. On the other hand, it is an established fact that DDT had been used extensively during a typhus outbreak in Japan and Korea in 1945 - 1946 and that DDT was subsequently available to the occupation forces.

Starting with a field-collected strain of lice, K. Yasutomi was able to select in three generations a resistant strain capable of withstanding residual deposits of 60 to 70 times as much DDT as the original strain.

This report covers, in part, the results of a cooperative project by the Communicable Disease Center, Public Health Service, U. S. Department of Health, Education, and Welfare, Savannah, Ga., and the laboratory of the Entomology Research Division, Agricultural Research Service, U. S. Department of Agriculture, Orlando, Fla., designed to determine some of the factors involved in the resistance of DDT of the original Korean strain of body lice which has been maintained under DDT selection at that laboratory for several years.

A conspicuous feature of the results obtained with topical application is the extremely slow rate of absorption of DDT through the louse cuticle. Owing to the particularly tough and oily nature of this cuticle, solvents such as ethanol, acetone, or benzene proved unsuitable for topical application. Some higher alcohols were too toxic, and several vegetable and mineral oils, including Risella oil, were found unsatisfactory.

Because no marked differences in absorption were found between resistant and susceptible lice, rate of penetration of DDT may be ruled out as a defense mechanism of the resistant strain. Also, it is evident that only minute amounts of DDT were needed internally to cause complete mortality of the susceptible strain.

Greater differences in tolerance for DDT are clearly demonstrated in the feeding experiments. The fact that resistant lice could tolerate 100 p. p. m. of DDT in the blood with minimum mortalities, whereas 10 p. p. m. were lethal to more than 50% of the susceptible lice, indicates the presence of an intrinsic biochemical difference and strengthens the conclusion that penetration of DDT through the cuticle is not an important factor.

The metabolism tests in vivo indicated that the ingested DDT was almost completely metabolized by the resistant strain, whereas little or no degradation of DDT occurred in the susceptible strain. While a concentration of 10 p. p. m. of DDT produced 53% mortality of the susceptible lice in 24 hours, the limit of sensitivity of the analytical methods did not permit the use of lower concentrations in order to ascertain whether susceptible lice could metabolize sublethal doses of DDT.

Although the metabolite obtained from the in vivo metabolism of DDT by the resistant strain has not been positively identified, certain of its properties have been clearly defined. On the basis of solubility characteristics, both before and after nitration, DDE (dichlorodiphenyl dichloroethylene) might be ruled out as a possible metabolite, although it is conceivable that conjugation with a protein or a lipotrotein might sufficiently alter the solubility of DDE to form a more polar conjugate.

In limited experiments, using topical applications of DDT to the same strain of resistant lice mentioned in this report, C. W. Kearns reported the presence of a metabolite that responded in the same manner as DDE to the Schechter-Haller colorimetric test. On the other hand, tests in vitro, using lice homogenates, showed no evidence of the existence of a mechanism comparable to that found in the resistant house fly. Kearns stated, however, that owing to difficulties encountered in extraction and removal of interfering materials, these findings were not conclusive.

It is worth noting that none of the synergists tested in combination with DDT appreciably increased the mortality of the resistant strain. These findings are in agreement with results of M. M. Cole who reported no increase in the mortality of DDT-resistant lice exposed to DDT : DMC (1, 1-bis-(p-chlorophenyl) methyl carbinol) and DDT : MR-60 (bis-(p-chlorophenyl) chloromethane) combinations. Other investigations have demonstrated that certain DDT-synergists, including those mentioned above, interfere with the enzymatic conversion of DDT to DDE in house flies both in vivo and in vitro. These results strengthen the assumption that the metabolite from lice is not DDE and that the mechanism of DDT-breakdown is different from that found in flies.

Experiments in vitro distinctly showed that homogenates and acetone powders of both resistant and susceptible lice are capable of metabolizing DDT at an approximately equal rate. By analogy with the enzyme DDT-dehydrochlorinase which is found in DDT-resistant house flies, but not in any measurable quantity in susceptible strains, one might infer that enzymatic breakdown of DDT in lice is a natural phenomenon, i. e., that all strains of lice, both resistant and susceptible, possess a "hidden resistance potential" to DDT in the form of a detoxifying enzyme system whose action on DDT is interfered with by some other unknown biochemical factor which is operative in the susceptible strain, but is bypassed in the resistant strain. This bypass mechanism which is accentuated through selection pressure with DDT would then allow the resistant louse sufficient survival time to enable the enzyme to neutralize the absorbed insecticide. In order to arrive at a logical conclusion, these hypotheses must be tested further by investigating several other susceptible and DDT-resistant strains from various localities. (Perry, A. S., Buckner, A. J., Biochemical Investigations on DDT-Resistance in the Human Body Louse, Pediculus humanus humanus: Am. J. Trop. M. Hyg., 7: 620-626, November 1958)

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Policy

The U. S. Navy Medical News Letter is basically an official Medical Department publication inviting the attention of officers of the Medical Department of the Regular Navy and Naval Reserve to timely up-to-date items of official and professional interest relative to medicine, dentistry, and allied sciences. The amount of information used is only that necessary to inform adequately officers of the Medical Department of the existence and source of such information. The items used are neither intended to be, nor are they, susceptible to use by any officer as a substitute for any item or article in its original form. All readers of the News Letter are urged to obtain the original of those items of particular interest to the individual.

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